SALIVARY LEVELS OF TUMOR NECROSIS FACTOR-ALPHA AND INTERLEUKIN- 32 ALPHA IN PERIODONTITIS: A CASE-CONTROL STUDY

SIVAN MOHAMMED HASAN*
HASHIM DAWOOD MOUSA**

Submitted 13 July 2025; accepted 23 December 2025; Published 01 July 2026

ABSTRACT

Background: Periodontitis is an inflammatory disease that causes progressive destruction of periodontal tissues. Cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-32 alpha (IL-32α) are believed to play key roles in its pathogenesis. This research aimed to evaluate the salivary levels of TNF-α and IL-32α among patients with different severities of chronic periodontitis and periodontally healthy individuals in Kirkuk City, Iraq.

Methods: This case-control study included 90 systemically healthy individuals aged between 20–55 years, divided into four groups: healthy controls, and patients with mild, moderate, and severe periodontitis, according to the 1999 American academy of periodontology (AAP) classification. Unstimulated saliva samples were collected and analyzed for Tumor necrosis factor-alpha (TNF-α) and Interleukin-32 alpha (IL-32α) concentrations using enzyme-linked immunosorbent assay (ELISA). Clinical parameters such as plaque index (PI), probing pocket depth (PPD), and clinical attachment loss (CAL) were recorded. Data were analyzed using Analysis of Variance (ANOVA), correlation analysis, and a receiver operating characteristic (ROC) curve assessment.

Results: Both TNF-α and IL-32α levels were significantly elevated in periodontitis patients compared to healthy individuals, with a progressive increase corresponding to disease severity. TNF-α showed strong correlations with clinical parameters and a higher diagnostic accuracy while IL-32α also increased with severity but had lower diagnostic performance.

Conclusion: Salivary TNF-α and IL-32α are elevated in chronic periodontitis and correlate with disease severity. TNF-α, in particular, has the potential to be a non-invasive biomarker for diagnosing and monitoring periodontal inflammation but further researches are required

Duhok Med J 2026; 20 (1): 24-35.

Keywords: Chronic periodontitis, Saliva, Cytokines, Tumor necrosis factor-alpha, Interleukin-32 alpha.

Periodontal diseases are degenerative, damaging, and inflammatory conditions affecting the tissues that support the teeth[1,2]. Periodontitis is believed to be initiated by dental biofilms that build up subgingivally at the gingival margin[3]. A range of chemokines, cytokines, and inflammatory mediators are generated by immune and inflammatory cells that target affected periodontal tissues[4]. Saliva has traditionally been seen as an indicator of overall bodily health, as it contains serum components used in conventional blood tests for disease and health monitoring. As a diagnostic fluid, saliva provides multiple advantages over serum for disease diagnosis[5]. Saliva collection is non-intrusive, hence easier, safer, and more cost-effective than blood collection. Saliva collection can be conducted without the help of trained personnel, in contrast to blood collection[6]. Tumor necrosis factor-alpha (TNF-α) is a proinflammatory cytokine with extensive effects, including tissue destruction and cell migration. It influences cell migration by upregulating

* *Dept of periodontics, College of dentistry, Duhok, Iraq. Email: sivanmuhammed9@gmail.com*
** *Dept of Periodontics, Assistant Dean of College of Dentistry, Duhok University, Mob:+967504711049*

https://doi.org/10.31386/dmj.2026.20.1.3

adhesion molecules, facilitating neutrophil rolling and adherence to the vascular wall, ultimately leading to extravasation[7]. It has been shown to activate matrix metalloproteinases (MMPs), which are implicated in the degradation of periodontal connective tissue during the advancement of periodontitis[8]. Moreover, TNF-α facilitates the secretion of MMPs and Receptor Activator of Nuclear Factor-kappa B Ligand (RANKL), which are associated with extracellular matrix degradation and bone resorption[9,10]. Interleukin-32 (IL-32) is a pro-inflammatory cytokine associated with numerous inflammatory diseases, including periodontal diseases. It plays a role in the etiology of periodontal diseases by regulating immune responses and enhancing inflammation. This cytokine is integral to a complex network of cytokines that regulate the inflammatory response in periodontal tissues, and its function is interconnected with other cytokines and immune cells participating in the disease process. Interleukin-32 (IL-32) is synthesized by multiple cell within the periodontium, such as fibroblasts and immune cells, and its expression is enhanced in reaction to bacterial components, including lipopolysaccharides (LPS) from periodontal pathogens[11] .IL-32 interacts with the IL-1 cytokine family, which are critical contributors to periodontal inflammation. The IL-1 family consists of both pro-inflammatory as well as anti-inflammatory cytokines, with IL-32 capable of modulating the equilibrium between these molecules[12].

The cytokine network in periodontitis is intricate, with IL-32 perhaps regulating the function of natural killer-like B cells and macrophages, which participate in the immune response to periodontal infections[11,13].

IL-32 may also affect the polarization of macrophages towards a pro-inflammatory M1 phenotype, which is linked to heightened tissue damage in periodontitis[11].

The study aims and objectives

This research aimed to evaluate the salivary levels of Tumor necrosis factor-alpha (TNF-α) and Interleukin-32 (IL-32α) among patients with different severities of chronic periodontitis and periodontally healthy individuals. Evaluate their relation with the disease conditions also to determine whether or not saliva might be a reliable medium to detect the severity of the disease condition.

MATERIALS AND METHODS:

Setting and time of the study

The present study was executed in kirkuk City, the study was conducted from November 2024 to October 2025. The current study received approval from the research ethical committee of the Duhok Directorate of Health and the College of Dentistry, University of Duhok (Reference number: 13042025-3-13). All participants received comprehensive information regarding the study's purpose and objectives. Subsequently, each participant in the study submitted a written consent form.

Sample size calculation

To calculate the sample size for each group, we used ANOVA considering both main effects and interactions. The study included four groups: healthy individuals and patients with mild, moderate, or severe chronic periodontitis. Effect size was determined using G*Power 3.1.9. For this calculation, TNF-α levels (in pg/mL) from five preliminary cases per group were used. The standard deviation (SD) within each group was 15.5 pg/mL, and the mean TNF-α values for the healthy, mild, moderate, and severe groups were 193.23, 198.40, 202.36, and 215.65 pg/mL, respectively. The initial sample size estimate was 20 participants per group. However, we increased this to 22 per group to account for potential dropouts or data exclusions. The effect size was 0.54, the significance level

(α) was set at 0.05, and the desired power was 0.95.

Study population and study design

The research design was a a case-control study aimed at evaluating the level of interleukin 32 and tumor necrosis factor alpha in the saliva of periodontitis patients and healthy individuals. The current study was conducted on a range of individuals with healthy periodontium (control group) to patients with severe periodontitis. Our study included 90 patients of both genders ranging in age from 20 to 55 years old. Patients were examined to determine disease condition and saliva samples collection were performed.

Inclusion and exlusion criteria

Healthy individuals and patients with a diagnosis of various severities of periodontitis depending on the criteria referred to in 1999 AAP classification of periodontal disease with the following characteristics; absence of systemic diseases , age within 20 to 55 years, and no scaling and root surface debridement or any form of periodontal therapy for at least six months. Exlusion criteria involves; patients who have undergone or are presently receiving substantial periodontal therapy, Patients who have used anti-inflammatory or antibiotic medications in the past three months, immunocompromised patients, Smokers, females who utilize contraceptive pills, postmenopausal females, lactating , and pregnant females[1,2,14].

Patient Groups

The study population was divided into the subsequent groups depending on the criteria referred to in 1999 AAP classification of periodontal disease[15]; group 1 (control group); Included 22 healthy periodontal individuals with no clinical signs of inflammation (no bleeding on probing, redness, or swelling), probing pocket depth (PPD): ≤3 mm , and no Clinical Attachment Loss (CAL). Group 2 Mild; 1 to 2 mm (CAL) , (PPD) 3–5 mm , 22 patients . Group 3 Moderate: 3 to 4 mm CAL, (PPD) 5–7 mm, 22 patients. Group 4 Severe, 24 patients (CAL) ≥5 mm, (PPD) ≥7 mm.

Saliva samples collection:

Whole unstimulated saliva (Five milliliters) were collected from the participating individuals. The samples were gathered in the morning [between 08.00 and 12.00], at least two hours postprandial. Unstimulated whole saliva was collected using the Navazesh method which dictates that Participants were instructed to avoid eating, drinking, smoking, or performing oral hygiene for at least 1–2 hours before sample collection. Sample Collection were performed in the morning with the participant seated upright with the head slightly tilted forward. Saliva was allowed to accumulate naturally in the mouth and expectorated into sterile tubes every 60 seconds over 5–15 minutes to obtain an adequate volume. Collected samples were immediately placed on ice and centrifuged at 3000 rpm for 15 minutes at 4°C to remove cellular debris and mucus. The resulting supernatant was stored at −20°C until analysis to preserve the stability of salivary cytokines[16]. And were used for laboratory analysis within a time not expanding more than six months from collection time since cyrokines stability are affected after that period[17].

Clinical measurements

An examination was conducted, encompassing the Plaque Index (PLI), Bleeding on Probing (BOP), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL). A periodontal probe (University of North Carolina (UNC 15) periodontal probe) was employed to assess all clinical periodontal parameters. Each tooth, excluding the wisdom teeth, was evaluated for six surfaces, four of which were designated for plaque assessment. The clinical examination was conducted by a single examiner for all subjects.

Laboratory procedure

The concentrations of TNF-α and IL-32-α were determined in the saliva of

periodontitis and control groups with an (ELISA) kit (sunlong biotech -china) in accordance with the manufacturer's standards. The absorbance was measured utilizing a spectrophotometer plate reader (Paramedical, Italy)

Statistical analyses

The age and gender of the healthy individuals and chronic periodontitis were presented in mean (SD) and no (%), respectively. The homogeneity of the study groups for the age and gender was examined in an independent t-test and Chi-square test, respectively. Comparisons of main biomarkers and TNF-α and IL-32 α among health individuals and patients with different chronic periodontitis were examined in ANOVA one-way test. The pairwise comparisons were examined in a Tukey test. The correlations of TNF-α and IL-32 α with main biomarkers in chronic periodontitis were determined in bivariate regression analysis. The sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy for the role of TNF-α and IL-32 α in the diagnosis of chronic periodontitis were determined in Roc curve analysis. The significant level of difference was determined in a $p<0.05$. The statistical calculations were performed using JMP®, Version 18.0. SAS Institute Inc., Cary, NC, 1989–2023.

RESULTS:

The study included 22 healthy individuals and 68 patients with chronic periodontitis . The mean age of the healthy group was 39.14 ± 9.34 years, while that of the periodontitis group was 36.44 ± 10.45 years. Statistical analysis showed no significant difference in age between the two groups ($p = 0.2840$). Regarding gender distribution, males represented 59.09% of the healthy group and 57.35% of the periodontitis group, while females accounted for 40.91% and 42.65%, respectively. The difference in gender distribution was not statistically significant ($p = 0.8859$). These findings indicate that both groups were comparable in terms of age and gender as shown table 1.

Table 1. General demographic and clinical characteristics of the study population.

Characteristics	Study groups		P value
	Healthy (n=22)	chronic periodontitis (n=68)	
age	39.14 (9.34)	36.44 (10.45)	0.2840
Gender: Male	13 (59.09)	39 (57.35)	0.8859
Female	9 (40.91)	29 (42.65)	

n: number of the subjects

Healthy individuals (n = 22) and patients with chronic periodontitis (n = 68). Data are presented as mean (SD) for age and gender. Statistical differences were tested using an independent t-test for age and Chi-square test for gender distribution

TNF-α levels increased progressively from healthy individuals (195.31 ± 15.00 pg/mL) to mild (201.37 ± 12.13 pg/mL), moderate (207.66 ± 15.40 pg/mL), and severe periodontitis (217.77 ± 14.58 pg/mL), with a statistically significant overall difference ($p < 0.0001$). Significant pairwise differences were observed between severe vs. healthy groups ($p < 0.0001$), severe vs. mild groups ($p = 0.0012$), and moderate vs. healthy groups ($p = 0.0270$), though differences between adjacent stages were not significant. Grouped analysis also showed significantly higher TNF-α levels in periodontitis patients compared to healthy controls ($p = 0.0004$).

Similarly, IL-32α levels rose with disease severity from 88.13 ± 17.15 pg/mL (control group) to 108.15 ± 19.74 pg/mL (severe group) with an overall significant difference ($p = 0.0017$). Only the severe vs. healthy groups comparison reached

statistical significance (p = 0.0007), while the group comparison also confirmed higher IL-32α levels in periodontitis cases (p = 0.0013). These findings suggest both biomarkers increase with disease severity, with TNF-α showing stronger discriminatory values as shown in table 2.

Table 2. Mean salivary concentrations of TNF-α and IL-32α among healthy individuals and patients with varying severities of chronic periodontitis, with intergroup comparisons using one-way ANOVA and Tukey's post-hoc test.

	Mean (SD)	P value	Pairwise comparisons
TNF-α (pg/mL)			Severe > healthy (p<0.0001)
Healthy	195.31 (15.00)	<0.0001	severe > mild (p=0.0012)
Mild	201.37 (12.13)		moderate > healthy (p=0.0270)
Moderate	207.66 (15.40)		moderate > mild (p=0.4685)
Severe	217.77 (14.58)		
chronic periodontitis	209.19 (15.51)	<0.0004	
healthy	195.310 (15.00)		
IL-32 α (pg/mL)		<0.0017	Severe > healthy (p=0.0007)
Healthy	88.13 (17.15)		
Mild	97.37 (14.14)		
Moderate	100.34 (15.65)		
Severe	108.15 (19.74)		
chronic periodontitis	102.14 (17.17)	<0.0013	
healthy	88.13 (17.15)		

*Data are expressed as mean (SD) in pg/mL. P values were obtained using one-way ANOVA with Tukey's post-hoc pairwise comparisons. Significant group differences are indicated. * IL: interleukin; TNF: tumor necrosis factor; pg/mL: picograms per milliliter.*

the diagnostic performance of salivary TNF-α and IL-32α in distinguishing chronic periodontitis and its severity across the groups was evaluated using a receiver operating characteristic (ROC) curve as shown in table 3. TNF-α showed the highest diagnostic accuracy in severe periodontitis, with 83.3% sensitivity, 86.4% specificity, and 84.78% accuracy at a cutoff of 207.41 pg/mL. Diagnostic performance declined in moderate (accuracy 68.18%) and mild cases (accuracy 59.09%). When all periodontitis cases were grouped, TNF-α demonstrated moderate diagnostic value (accuracy 67.78%, cutoff 202.85 pg/mL).

IL-32α also performed best in severe cases (accuracy 78.26%, cutoff 99.2 pg/mL), with moderate effectiveness in moderate (68.18%) and mild (63.64%) periodontitis. For all cases combined, IL-32α showed moderate diagnostic accuracy (67.78%, cutoff 94.87 pg/mL).

In summary, both biomarkers showed increasing diagnostic value with disease severity, particularly in severe periodontitis, but limited effectiveness in detecting mild and moderate cases.

Table 3. Diagnostic performance of salivary TNF-α and IL-32α in distinguishing chronic periodontitis and its severity levels from healthy individuals, based on ROC curve analysis.

Marker	Comparison	Cuttoff (pg/ml)	Sensitivity (%)	Specificity (%)	Accuracy (%)
TNF-α	Chronic Periodontitis vs. healthy	202.85	66.2	72.7	67.78
TNF-α	Severe vs. healthy	207.41	83.3	86.4	84.78
TNF-α	Moderate vs. healthy	203.28	63.6	72.7	68.18
TNF-α	Mild vs. healthy	202.85	45.5	72.7	59.09

Marker	Comparison	Cuttoff (pg/ml)	Sensitivity (%)	Specificity (%)	Accuracy (%)
IL-32 α	Chronic Periodontitis vs. healthy	94.87	67.6	68.2	67.78
IL-32 α	Severe vs. healthy	99.2	75.0	81.8	78.26
IL-32 α	Moderate vs.healthy	94.94	68.2	68.2	68.18
IL-32 α	Mild vs. healthy	94.12	63.6	63.6	63.64

Diagnostic performance of salivary TNF-α and IL-32α in differentiating chronic periodontitis (overall and by severity) from healthy individuals, based on receiver operating characteristic (ROC) curve analysis. Cutoff values (pg/mL), sensitivity, specificity, and accuracy are reported. *Abbreviations: IL, interleukin; TNF, tumor necrosis factor ,pg/mL: picograms per milliliter.*

DISCUSSION:

Periodontitis is a chronic inflammatory condition driven by dysbiotic microbiota and mediated by an exaggerated host immune response. Among the inflammatory mediators are tumor necrosis factor-alpha (TNF-α) and interleukin-32 alpha (IL-32α) which the later have been recently recognized and both play a role in promoting tissue destruction and immune activation[4,18]. In this study, we observed a progressive and statistically significant elevation of salivary TNF-α and IL-32α levels from control group to those with mild, moderate, and severe chronic periodontitis groups. Importantly, our ROC curve analysis provided insight into the diagnostic performance of these cytokines. TNF-α demonstrated its strongest discriminative ability in distinguishing severe periodontitis from periodontal health condition. IL-32α also showed an increasing trend with disease severity, but overall performed less strongly than TNF-α.

Several previous studies support these findings . Layedh et al. (2021)[19] found that salivary TNF-α was significantly elevated in chronic periodontitis patients compared to healthy controls. Pulivarthi et al. (2022)[20], also demonstrated high baseline TNF-α levels in periodontitis, which decreased significantly after non-surgical therapy, indicating its responsiveness to inflammatory burden. However, some studies have presented contrasting results. Kim and Kim (2021)[21], in a meta-analysis, noted higher TNF-α in healthy controls and post-treatment cases, which may reflect variations in sampling techniques or study populations rather than cytokine behavior alone. Eivazi et al. (2017)[22], found reduced TNF-α levels in periodontitis cases. Such inconsistencies likely reflect differences in disease stage, patient selection, and methodological factors rather than true biological contradiction, but they highlight the complexity of cytokine regulation.

With respect to IL-32α, our study also showed significantly elevated salivary concentrations in periodontitis patients, consistent with the series of investigations by Onguzdede[23] and colleagues. Their early work demonstrated increased IL-32 in gingival crevicular fluid and saliva of chronic periodontitis patients, while their later study (2022)[18] revealed elevated IL-32 expression in gingival tissues and serum, indicating both local and systemic involvement. In a further related study, they again confirmed stronger IL-32 expression in inflamed periodontal tissues compared with healthy controls. These findings are supported by Dede et al. (2017)[24], who noted positive correlations between IL-32 and TNF-α, suggesting synergistic roles in inflammation. However, not all studies have the same conclusions Faria et al. (2021)[25], who examined IL-32 in gingival crevicular fluid, reported no significant differences between healthy and diseased groups, which may be due to differences in

fluid type, smaller subgroup sizes, and inclusion of implant-related conditions. More recently, Relvas et al. (2024)[16] suggested that other cytokines, such as IL-1β and IL-6, may sometimes correlate more strongly with disease activity than TNF-α or IL-32, reflecting the multifactorial nature of the periodontal inflammatory network.

Taken together, the majority of published evidence, including our findings, indicates that TNF-α and IL-32α are elevated in periodontitis cases compared to healthy control groups and contribute to its inflammatory pathogenesis. However, given the inconsistencies across studies and the modest diagnostic accuracy shown in our ROC analysis, it would be premature to regard them as dependable biomarkers. Their role may lie in serving as adjunctive salivary indicators of disease activity, particularly for more advanced cases.

Limitations

This study is limited by its modest sample size, single-center recruitment, case-control design and reliance on saliva alone without parallel GCF or serum measurements. In addition, cytokine stability may have been influenced by storage and handling conditions. These factors restrict the generalizability of our results, and longitudinal, multi-center studies with larger cohorts are needed to confirm whether salivary TNF-α and IL-32α can serve as reliable adjunctive markers for disease monitoring.

CONCLUSION

This study demonstrated that salivary levels of TNF-α and IL-32α are significantly elevated in individuals with chronic periodontitis and progressively increase with disease severity. TNF-α showed strong and consistent correlations with clinical periodontal parameters, highlighting its potential as a non-invasive biomarker for diagnosing and monitoring periodontal disease. IL-32α, while showing moderate diagnostic performance, was positively associated with TNF-α and clinical indicators, suggesting a contributory role in periodontal inflammation.

These findings show the potential role of saliva as a diagnostic fluid and the use of inflammatory cytokines, particularly TNF-α, in assessing periodontal disease status. The integration of salivary biomarkers into periodontal evaluation might improve early detection, however further longitudinal and mechanistic studies are warranted to explore the dynamic behavior of these cytokines during disease progression and therapy. Incorporating larger, more diverse populations and combining multiple biomarkers may enhance diagnostic precision and contribute to the advancement of personalized periodontal care.

Recommendations for Future Studies

Future research should consider larger and more diverse populations to enhance the generalizability of the findings. Longitudinal studies are recommended to better understand the dynamic changes of IL-32α and TNF-α levels throughout the progression and treatment of periodontitis. Additionally, future studies could explore the relationship between these salivary biomarkers and other systemic inflammatory markers to investigate possible links between periodontitis and systemic diseases. Using more sensitive techniques or combining saliva analysis with gingival crevicular fluid assessment might improve biomarker accuracy. Finally, experimental studies could examine the role of IL-32α in periodontal tissue destruction to identify potential therapeutic targets.

Conflict of Interest:

The authors declare no conflict of interest related to this study.

Acknowledgment and Funding:

The authors thank the College of Dentistry, University of Duhok, for their support. No specific funding was received for this study.

the article under the journal's open-access policy.

REFERENCES:

1. 1. Mohammed HA, Abdulkareem AA, Zardawi FM, Gul SS. Determination of the accuracy of salivary biomarkers for periodontal diagnosis. Diagnostics. 2022;12(10):2485. doi:10.3390/diagnostics12102485.
2. 2. Saliem SS, Bede SY, Cooper PR, Abdulkareem AA, Milward MR, Abdullah BH. Pathogenesis of periodontitis: a potential role for epithelial–mesenchymal transition. Jpn Dent Sci Rev. 2022;58:268–78. doi:10.1016/j.jdsr.2022.05.002.
3. 3. Hajishengallis G. Periodontitis: from microbial immune subversion to systemic inflammation. Nat Rev Immunol. 2015;15(1):30–44. doi:10.1038/nri3785.
4. 4. Neurath N, Kesting M. Cytokines in gingivitis and periodontitis: from pathogenesis to therapeutic targets. Front Immunol. 2024;15:1435054. doi:10.3389/fimmu.2024.1435054.
5. 5. Lee J, Lee JB, Song HY, Son MJ, Li L, Rhyu IC, et al. Diagnostic models for screening of periodontitis with inflammatory mediators and microbial profiles in saliva. Diagnostics. 2020;10(10):820. doi:10.3390/diagnostics10100820.
6. 6. Ligtenberg AJM. Saliva diagnostics: a summary of the expanded possibilities. Ned Tijdschr Tandheelkd. 2020;127(10):561–6. doi:10.5177/ntvt.2020.10.20034.
7. 7. Aguilar G, Córdova F, Koning T, Sarmiento J, Boric MP, Birukov K, et al. TNF-α-activated eNOS signaling increases leukocyte adhesion through the S-nitrosylation pathway. Am J Physiol Heart Circ Physiol. 2021;321(6):H1083–95. doi:10.1152/ajpheart.00065.2021.
8. 8. Ena S, Radhika G, Sreejith K, Ruhee S, Simaran T, Maninder K. Role of MMPs in connective tissue breakdown and periodontal disease: a review. Int J Recent Innov Med Clin Res. 2024;6(4):103–7. doi:10.18231/j.ijrimcr.2024.059
9. 9. Kim H, Kim E, Park AK, Shin Y, Kang J, Lim J, et al. Detection of association between periodontitis and polymorphisms of IL-1β+3954 and TNF-α-863 in the Korean population after controlling for confounding risk factors. J Periodontal Res. 2020;55(6):905–17. doi:10.1111/jre.12783.
10. 10. Jain P, Ved A, Dubey R, Singh N, Parihar AS, Maytreyee R. Comparative evaluation of serum tumor necrosis factor α in health and chronic periodontitis: a case–control study. Contemp Clin Dent. 2020;11(4):342–9. doi:10.4103/ccd.ccd_244_20.
11. 11. Pathak JL, Fang Y, Chen Y, Ye Z, Guo X, Yan Y, et al. Downregulation of macrophage-specific act-1 intensifies periodontitis and alveolar bone loss possibly via TNF/NF-κB signaling. Front Cell Dev Biol. 2021;9:628139. doi:10.3389/fcell.2021.628139.
12. 12. Papathanasiou E, Conti P, Carinci F, Lauritano D, Theoharides TC. IL-1 superfamily members and periodontal diseases. J Dent Res.

2020;99(13):1425–34. doi:10.1177/0022034520945209.

13. 13. Zhang Y, Kuang W, Li D, Li Y, Feng Y, Lyu X, et al. Natural killer-like B cells secreting interleukin-18 induce a proinflammatory response in periodontitis. Front Immunol. 2021;12:641562. doi:10.3389/fimmu.2021.641562
14. 14. Hassan ZM, Akram HM. Salivary biomarkers of inflammasome activation in unstable periodontitis: a case–control study. Eur J Dent. 2025. doi:10.1055/s-0045-1806931.
15. 15. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol. 1999;4(1):1–6. doi:10.1902/annals.1999.4.1.1.
16. 16. Relvas M, Mendes-Frias A, Gonçalves M, Salazar F, López-Jarana P, Silvestre R, et al. Salivary IL-1β, IL-6, and IL-10 are key biomarkers of periodontitis severity. Int J Mol Sci. 2024;25(15):8401. doi:10.3390/ijms25158401.
17. 17. Chauhan M. Storage of saliva and blood specimen at different temperatures. Int J Forensic Med. 2020;2(2):21–4. doi:10.33545/27074447.2020.v2.i2a.28.
18. 18. Öngöz Dede F, Gökmenoğlu C, Deveci ET, Çelen S, Avci B, Kara C, et al. Increased levels of interleukin-32 isoforms alpha, beta, gamma, and delta in the gingival crevicular fluid and plasma of patients with periodontitis. J Periodontal Res. 2021;56(1):83–92. doi:10.1111/jre.12796.
19. 19. Layedh NH, Al-Rubbaey YA, Fahad AH, Al-Roubaey DAA. Salivary IL-6 and TNF-α in patients with periodontitis. Ann Trop Med Public Health. 2021;24(3):317–25. doi:10.36295/asro.2021.24546.
20. 20. Pulivarthi P, Chava VK, Gunupati S. Salivary tumor necrosis factor-alpha levels in periodontitis associated with diabetes mellitus after low-level laser therapy as an adjunct to scaling and root planing: a randomized clinical trial. J Indian Soc Periodontol. 2022;26(3):236–44. doi:10.4103/jisp.jisp_149_21.
21. 21. Kim JY, Kim HN. Changes in inflammatory cytokines in saliva after non-surgical periodontal therapy: a systematic review and meta-analysis. Int J Environ Res Public Health. 2021;18(1):194. doi:10.3390/ijerph18010194.
22. 22. Eivazi M, Falahi N, Eivazi N, Eivazi MA, Raygani AV, Rezaei F. The effect of scaling and root planing on salivary TNF-α and IL-1α concentrations in patients with chronic periodontitis. Open Dent J. 2017;11:573–8. doi:10.2174/1874210601711010573.
23. 23. Öngöz Dede F, Balli U, Bozkurt Doğan Ş, Güven B. Interleukin-32 levels in gingival crevicular fluid and saliva of patients with chronic periodontitis after periodontal treatment. J Periodontal Res. 2017;52(3):397–407. doi:10.1111/jre.12404.
24. 24. Dede FO, Balli U, Durmuslar MC, Doğan SB, Avci B, Ayas B, et al. Evaluation of IL-32 levels in gingival tissue and serum of an experimental periodontitis model. J Turgut Ozal

Med Cent. 2017;24(3):---. doi:10.5455/jtomc.2017.01.012.

25. 25. de Faria JB, de Souza Furtado TC, de Assunção TSF, Abdalla DR, Andrade FM, Bertoldo BB, et al. Immunological evaluation of the crevicular fluid in patients with gingivitis, periodontitis, and peri-implantitis: a 1-year cross-sectional study. Res Soc Dev. 2021;10(13):e41101320756. doi:10.33448/rsd-v10i13.20756.

پوختە

ئاستێن فاکتەرێ مرنا شانا دناف وەرمینێ دا ئەلفا و ئینتەرلێوکین 32 ئەلفا دناڤ تفێ دا لهەودانا پالپشتێن ددانی : خاندنەکا بواری کونترۆلکرنێ دا

پێشەکی و ئارمانج: هەودانا پالپشتێن ددانی نەخۆشیەکا هەودانێ یە و دبیتە ئەگەرێ وێ چەندێ کو پیچ پیچە شانەیێن پالپشتێن ددانی پوپچ ببن. دوێ باوەرێدایە کو سیتوکینات، وەك هۆکارێ رزیبوونا وەرەما ئەلفا (TNF-α) و ئەنترلوکین - 32 ئەلفا (IL - 35α) رۆلەکێ سەرەکی دگێریت بو تووشبوون ب ڤێ نەخۆشیێ. ئارمانجا ڤێ ڤەکولینێ هەلسەنگاندنا ئاستێن خوزیا (TNF-α) و (IL - 35α) یە لنك وان نەخۆشێن تووشی پلەیێن جیاوازبووین ژ هەودانا دوم درێژ یا پالپشتێن ددانی ل باژێرێ کەرکووك -عێراق.

رێکێن ڤەکولینێ: ئەڤێ ڤەکولینا برگەیی (90) کەسێن ساخلەم ب ئامیری بخوڤەگرت کو تەمەنێ وان دناڤبەرا 20 - 55 ساليێدا، و ل سەر چوار کۆمەلاندا هاتینە دابەشکرن: کۆمەلەکا راگرا ساخلەم، و کۆمەلەکا نەخۆشان تووشی هەودانا پالپشتێن ددانی یا سڤك یان ناڤنجی یان توند بووینە. لدویف پۆلینکرنا کۆمەلا ئەمریکی یا نوژداریا ددانان (AAP) یا سالا 1999 هندەك خوزیێن نەهاندەر کومکرینە و شرۆڤەکرن بو دەستنیشانکرنا چریێن TNF-α و IL - 32α بکارئینانا تاقیکرنا ئەلیزا (پیڤەرێ هەلمژینا بەرگریا دەرهاڤێژی) و پیڤەرێن تەختەبەندی تۆمارکرن، وەك نیشاندەرێ تابلۆیا تەمەنی، و کویراتیا بەرویکا هەستیاری، و ژ دەستدانا هەڤبەندیا تەختەبەندی، و داتا هاتنە شرۆڤەکرن بکارئینانا شرۆڤەکرنا جیاواز (ANOVA) و شرۆڤەکرنا هەڤبەندیێ و هەلسەنگاندنا چەماوێ ROC.

ئەنجام: ئاستێن هەر ئێك ژ هۆکارێ رزیبوونا وەرەما ئەلفا (TNF – α) و هۆکارێ ئەنترلوکین 32 ئەلفا (IL-32α) ب شێوەکێ بەرچاڤ بلندبوون لنك نەخۆشێن هەودانا پالپشتێن ددانی ب بەراوردکرن دگەل کەسێن ساخلەم، دگەل زێدەبوونەکا پیچ پیچە کو دگەل توندیا نەخۆشیێ دگونجیت. هۆکارێ رزیبوونا وەرەما ئەلفا (TNF – α) هەڤبەندیەکا بهێز دیارکر ب پیڤەرێن تەختەبەندی و هویریا دەستنیشانکرنەکا بلندتر، ب تایبەت د بوارێن توندا (84,78 %) هەروەسا ئاستێ هۆکارێ رزیبوونا وەرەما ئەلفا (IL - 32α) دگەل توندیا نەخۆشیێ بلندبوو، لێ دگەل کێمبوون د پێرابوونا وی یا دەستنیشانکرنێ.

دەرئەنجام: ئاستێ TNF – α و IL - 32α د خوزیا هەودانا پالپشتێن ددانی یێن دوم درێژ بلند دبیت و هەڤبەندی ب توندیا نەخۆشیێڤە هەیە. ب شێوەکێ تایبەت TNF – α دشێت وەك نیشاندەرەکێ کاریگەرێ نە نشتەرگەری یێ باوەرپێکری بیت بو دەستنیشانکرن و چاڤدێریکرنا هەودانا پالپشتیێن ددانی، و ئاموژگاری ئەوە کو گەلەك ڤەکولین بهێنەکرن بو دیارکرنا مفایێن وان دناڤ جورێن خەلکەکێ بەرفرەهدا و ل ژینگەکێن ماوەدرێژ.

الخلاصة

المستويات اللعابية لعامل نخر الورم ألفا والإنترليوكين-32 ألفا في التهاب انسجة ما حول الاسنان : دراسة حالات وشواهد

الخلفية والأهداف: التهاب دواعم السن هو مرض التهابي يسبب تدميرا تدريجيا لأنسجة دواعم السن. يعتقد أن السيتوكينات، مثل عامل نخر الورم ألفا (TNF-α) وإنترلوكين-32 ألفا (IL-32α)، تلعب دورا رئيسيا في التسبب بهذا المرض. هدفت هذه الدراسة إلى تقييم مستويات اللعاب لـ TNF-α وIL-32α لدى مرضى يعانون من درجات متفاوتة من التهاب دواعم السن المزمن وافراد اصحاء في مدينة كركوك، العراق.

الطرق: شملت هذه الدراسة المقطعية 90 فردا سليما جهازيا، تتراوح أعمارهم بين 20 و55 عاما، مقسمين إلى أربع مجموعات: مجموعة ضابطة صحية، ومجموعة مرضى يعانون من التهاب دواعم السن الخفيف، او المتوسط، او الشديد، وفقا لتصنيف الجمعية الأمريكية لطب الأسنان (AAP) لعام 1999. جمعت عينات لعاب غير محفزة، وحللت لتحديد تركيزات TNF-α وIL-32α باستخدام اختبار الإليزا (المقايسة الامتصاصية المناعية الإنزيمية). وسجلت المعايير السريرية، مثل مؤشر اللويحة السنية، وعمق الجيب المسباري، وفقدان الارتباط السريري. وحللت البيانات باستخدام تحليل التباين (ANOVA)، وتحليل الارتباط، وتقييم منحنى ROC.

النتائج: ارتفعت مستويات كل من عامل نخر الورم ألفا (TNF-α) وعامل إنترلوكين 32 ألفا (IL-32α) بشكل ملحوظ لدى مرضى التهاب دواعم السن مقارنة بالأفراد الأصحاء، مع زيادة تدريجية تتوافق مع شدة المرض. أظهر عامل نخر الورم ألفا (TNF-α) ارتباطا قويا بالمعايير السريرية ودقة تشخيصية أعلى، خاصة في الحالات الشديدة (84.78%). كما ارتفع مستوى الانترليوكين ٣٢ ألفا (IL-32α) مع شدة المرض، ولكن مع انخفاض في أدائه التشخيصي.

الاستنتاجات: يرتفع مستوى TNF-α وIL-32α في اللعاب في التهاب دواعم السن المزمن، ويرتبطان بشدة المرض. ويمكن لـ TNF-α، على وجه الخصوص، أن يكون بمثابة مؤشر حيوي غير جراحي موثوق لتشخيص ومراقبة التهاب دواعم السن. ويوصى بإجراء المزيد من الأبحاث لاستكشاف فائدتهما في فئات سكانية أوسع وفي بيئات طويلة الأمد.